MONITORING IMPROVEMENT AFTER VESTIBULOPATHY

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Images courtesy of Dr Jorge Kattah & Dr Nicolas Perez

Symptoms that occur after vestibulopathy

Unilateral central and peripheral vestibulopathies cause two types of symptoms and signs that are actively compensated over time:

Static symptoms are present when the head is still and they are typically associated with the acute phase of the lesion. The symptoms diminish after several days through the process of static compensation.

- Vertigo
- Spontaneous nystagmus
- Imbalance/Postural unsteadiness
- Nausea
- Vomiting

Dynamic symptoms are present when the head is moving and they typically become noticeable after static symptoms subside. Dynamic symptoms usually diminish within several days or weeks through the process of dynamic compensation. The recovery of vestibular symptoms is due to the neurophysiologic process of "vestibular compensation". About 20-30 percent of patients with a unilateral vestibular disorder have persistent symptoms.[6]

- Reduced vestibulo-ocular gain for head turns toward the affected side
- Loss of visual acuity during head movements
- Imbalance - veers from a straight path when walking
- Head-shake nystagmus

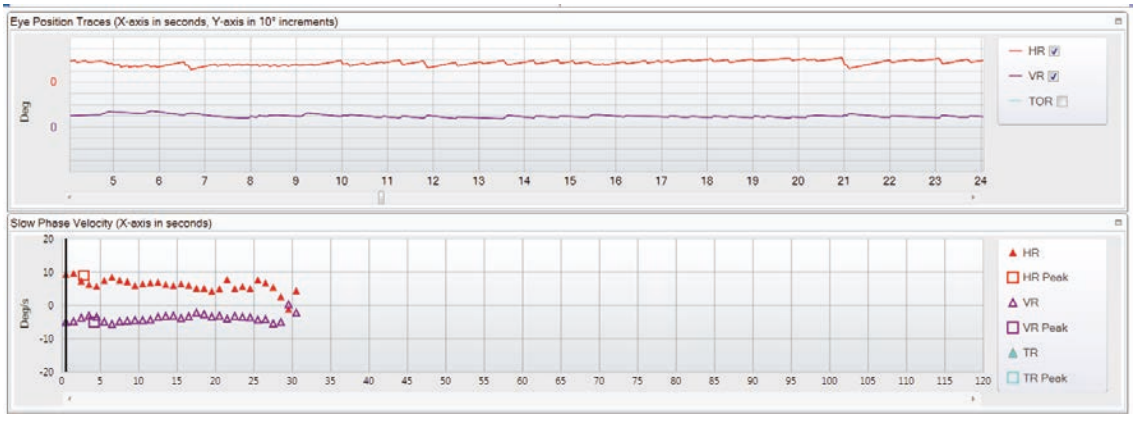
Changes in vestibular tests with compensation

Vestibular compensation is the general recovery of vestibular function with the passage of time and associated with central changes. Knowledge about the status of vestibular compensation is important for the management of patients with vestibular disorders. The following are findings in vestibular tests that change with compensation.

Spontaneous Nystagmus:

Intensity of vestibular nystagmus will reduce as compensation occurs

Day One: Spontaneous Sitting with Vision Denied 7 deg/sec left beating and 5 deg/sec downbeating (note: patient had vestibular neuritis)
HR = Horizontal Right, VR = Vertical Right



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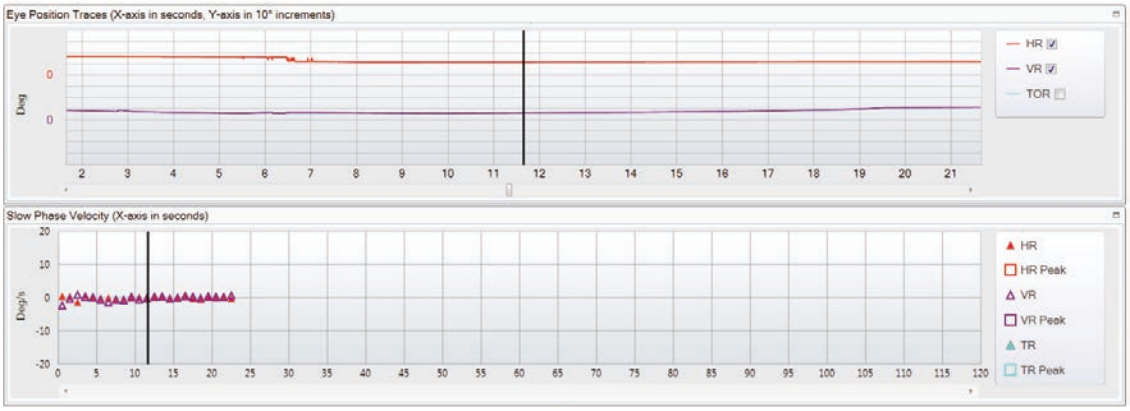
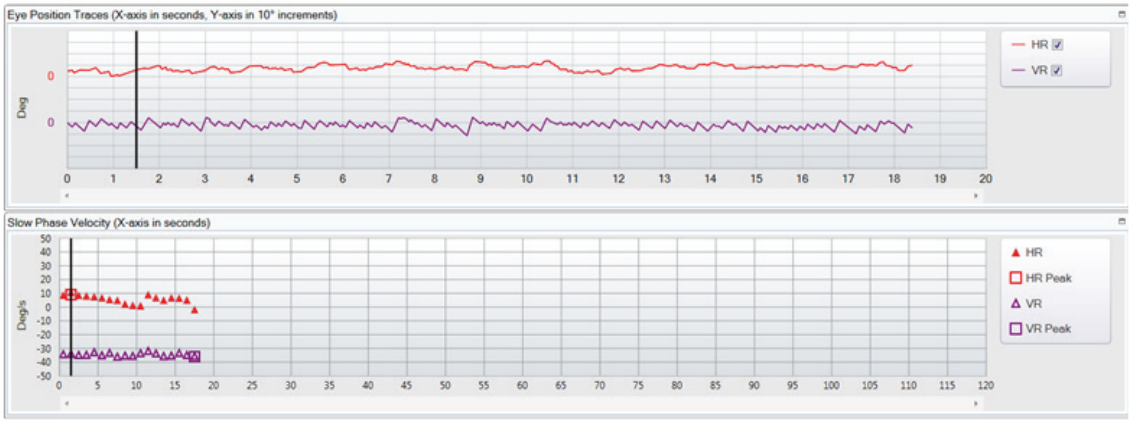
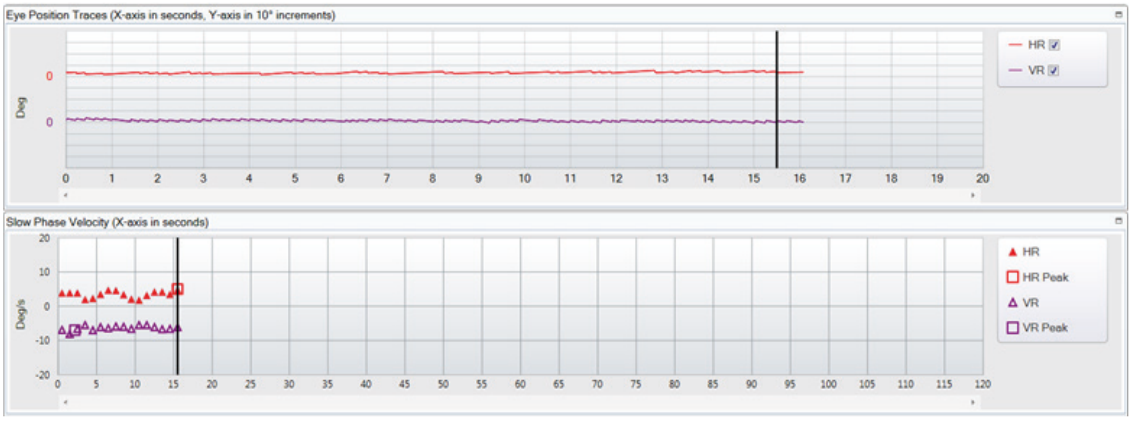
Day Three: Spontaneous Sitting with Vision Denied no nystagmus present



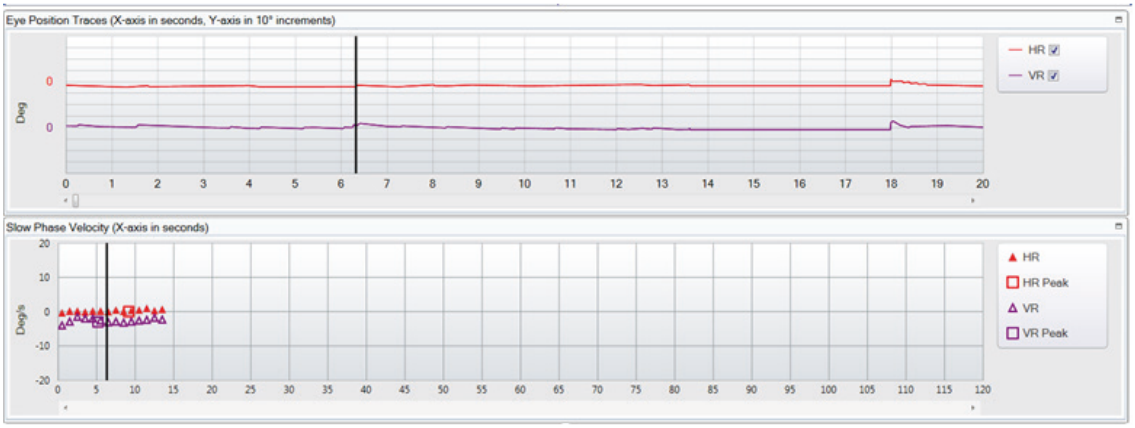
Day One: Gaze Center Sitting with Vision 9 deg/sec left beating and 36 deg/sec upbeating (note: this patient had a central pathology)



Day Three: Gaze Center Sitting with Vision 5 deg/sec left beating and 7 deg/sec upbeating

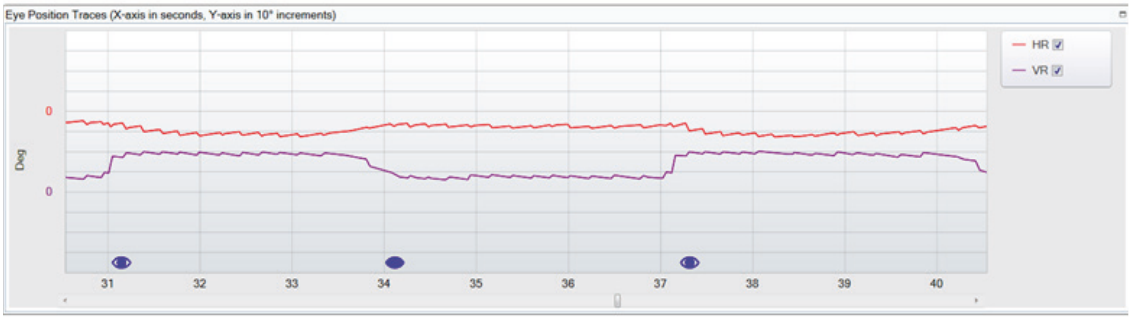


Day Seven: Gaze Center Sitting with Vision 0 deg/sec left beating and 3 deg/sec upbeating

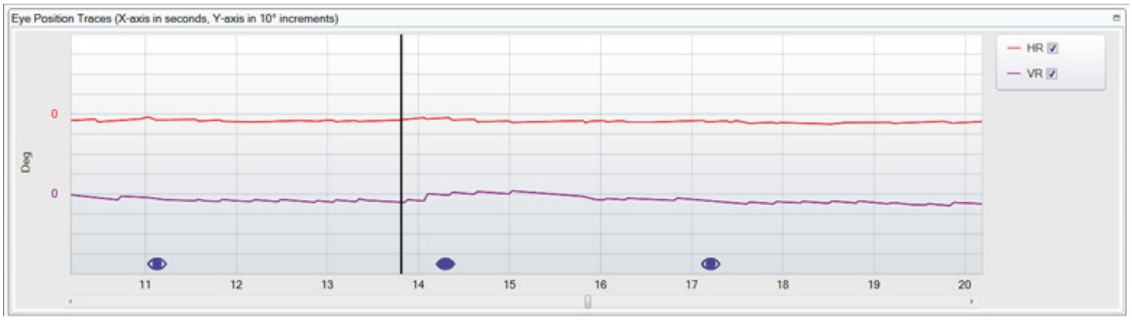


Skew Deviation: Average eye position shift will reduce as compensation occurs

Day One & Three: When the eye is covered there is 4 degrees horizontal rightward eye shift (right hypotropia) and 6.8 degrees vertical downward eye shift



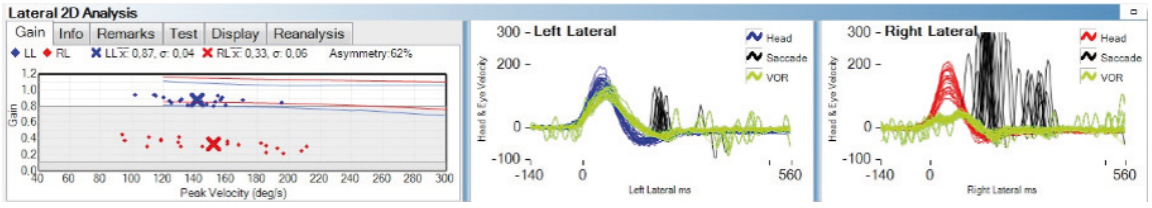
Day Seven: 0.8 degrees horizontal eye shift and 3.2 degrees vertical eye shift



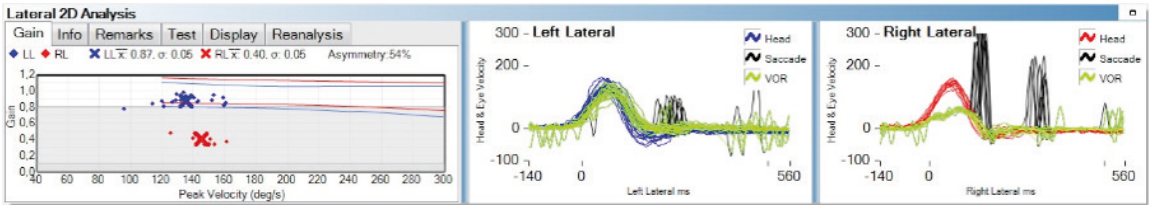
Catch-up Saccades during video Head Impulse Test (vHIT): Vestibular restoration is when the VOR for the ipsilesional head impulses fully recover meaning that gain is within normal limits and catch-up saccades are not present. This has been reported in patients with vestibular neuritis (Manzari et al. 2011[11]). Vestibular compensation can occur even when the function is not restored to the vestibular system. The latency of the catch-up saccades decrease and the presence of saccades change from overt to covert saccades as compensation occurs. There have been observed cases in which gain returns to within normal limits but overt saccades are well recorded. "The covert saccade acts to minimize the effect of the unilateral vestibular loss on the patients' permanent dynamic vestibulo-ocular reflex deficit because the saccade minimizes retinal smear, and saccadic suppression further reduces the perceptual experience" (MacDougall & Curthoys, 2012[10]). The patient who is "poorly compensated" is less likely to consistently produce covert saccades during video head impulse testing. Batuecas-Caletrio et al reported long-term follow-up after vestibular schwannoma surgery has shown that 22% of the patients display a particular abnormality in the VOR because refixation saccades occur in a random fashion after elicitation of the reflex in the HIT test. These patients report a higher level of vestibular disability and handicap.[2]

Video Head Impulse Test (vHIT): Catch-up saccades may move from overt to covert

Early Vestibular Neuritis: Right Lateral exhibits scattered overt and covert catch-up saccades around 200 to 400 msec
Left lateral exhibits overt saccades which can occur on the contralesional side. It is not uncommon to see a 10% or so reduction in contralesional VOR gain



Late Vestibular Neuritis: Right Lateral exhibits gathered covert and overt catch-up saccades with covert saccades around 90 to 140 msec



As reported by Schubert et al[14] "Covert (i.e. compensatory) saccades had a strong influence on reducing gaze position error associated with active and passive head impulses." Note active head impulse is when the patient moves their head and passive head impulse is when the tester moves the patient's head.

Gaze position error at the end of the ipsilesional active and passive head impulse		
	VOR With Covert	VOR Without Covert
Unilateral Vestibular Hypofunction		
Active	3.4 +/- 1.6	9.3 +/- 4.1
Passive	5.9 +/-2.7	9.1 +/-2.4
Bilateral Vestibular Hypofunction		
Active	4.3 +/-1.9	NA (fewer than 2 trials)
Passive	7.1 +/-3.0	6.7 +/- 1.1

This table was recreated with Dr Schubert's permission.[14]

This table is plotting gaze position error (GPE) a measure of the difference in eye position and target position after the head has stopped. The lower the number, the better the GPE, and thus 0 indicates no error. VOR with/without covert saccade refers to the contribution of the VOR and covert saccade to move the eyes and thus reduce the error. Without covert saccades, the GPE is greater (worse) in unilateral vestibular hypofunction (active or passive HIT). In bilateral vestibular hypofunction, there is a significant difference in GPE between active and passive only for VOR with covert saccades.

Rehabilitation:
Vestibular physical therapy instructs patients in home exercise routines that improve gaze instability (and postural instability). The gaze stability exercises are designed to challenge the brain to keep the eyes still in space during head rotation, when the vestibulo-ocular reflex (VOR) is deficient in doing so. Evidence suggests that this occurs from two means: an increase in the vestibular generated slow phase eye velocity (VOR gain) and an increase in the frequency of covert saccades

More Research needed
With the development of the video head impulse test and its widespread availability, we expect to learn more about the critical role of saccades and vestibulo-ocular gain on vestibular compensation. What triggers the patient to produce covert saccades? Why do some patients naturally produce them and compensate easily while others do not and have continued difficulty with their disorder? How can vestibular rehabilitation assist "poorly compensated" patients to learn how to produce covert saccades? Do all patients who produce covert saccades compensate well for their disorder?

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